right. In some cases it progresses to asthma as well. The inhalation challenge tests described reveal one factor linking the two disorders together.

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Anaphylaxis From Foods

IMMEDIATE SYSTEMIC ANAPHYLACTIC reactions to foods are caused by IgE-mediated hypersensitivity to a food allergen. Such reactions are serious, even life-threatening, and are not rare. The reaction occurs each time the food is eaten, even if the quantity ingested is minute. In some patients, handling the food or inhaling fumes while cooking may trigger an attack. In a patient with anaphylactic food sensitivity, there may be no other manisfestations of allergy, although the coexistence of an atopic history is common. Nuts, eggs, legumes, seafoods and berries are among the more common offenders, but any food is potentially capable of inducing sensitivity. Fortunately most patients with this disease report difficulty with only a single food or a group of related and allergenically cross-reacting foods, such as legumes.

The diagnosis is made in most cases by history. Because of the rapid onset of symptoms after ingestion of the food, the cause of the reaction is obvious to the patient. Sometimes, however, the association with meals is clear, but identification of the specific offending food is in doubt. In these cases skin testing for immediate wheal and flare responses to the suspected foods is appropriate. Because of the risk of a systemic reaction to the skin test, scratch or prick testing should be done first, and only if negative should this be followed by intradermal testing. The skin test is almost always positive, provided that the testing material is fresh. In some circumstances the physician may elect not to test because of fear of reaction to the test or for other reasons. A radioallergosorbent test (RAST) using the suspected food allergen then would be appropriate, since anaphylaxis to foods generally is associated with significant circulating IgE antibodies. The test is almost always positive in cases of anaphylaxis, bronchospasm or angioedema following ingestion of the food.

Occasionally an apparent reaction to a food is caused by a nonfood allergen. Additives, such as preservatives and coloring agents, are potentially allergenic. Penicillin present in milk and milk products has been responsible for systemic reactions in a few patients highly sensitive to the drug.

Specific diagnosis is mandatory for a patient who presents with a history of anaphylaxis after ingesting food, so that the patient can avoid future reactions. There is no evidence at present that oral or parenteral desensitization is effective.

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Human Breast Milk as a Protective Biological Fluid

HUMAN BREAST MILK is an ideal source of nutrition for infants. Although it has been known for some time that babies fed breast milk for the first 9 to 12 months have significantly less morbidity and mortality than infants fed cow's milk, characterization of host defense mechanisms passively imparted to immature infants has only recently been accomplished. Nonimmunological substances found in breast milk include a growth factor for actobacillus bifidus, which lowers the gut pH to decrease the survival of pathogenic organisms; lysozymes; lactoperoxidase, and lactoferritin—all of which destroy and inhibit bacterial growth. The major immunoglobulin in breast milk is secretory IgA which is critical for inhibition of macromolecule (foods and microbe) absorption in the gut. Specific antibody activity against Gram-positive and Gram-negative bacteria as well as viruses has been shown. Both IgG and IgE antibodies are not prominent in breast milk. Several complement components, particularly C3 and C4, also are found. Macrophages make up 90 percent of the cells found in breast milk, while lymphocytes constitute 10 percent of the cells. Both IgA secreting bone marrow derived B lymphocytes and functional thymus derived T cells are present. The protective role of the cellular elements in breast milk has not been elucidated, but is being actively studied.

Finally, human breast milk proteins do not appear to be allergenic to infants in contrast to the potentially allergenic bovine proteins found in

cow's milk. An infant is susceptible to absorption of macromolecules (both microbes and food antigens) in the first year of life. Breast milk may provide this immature host with passive protection at this critical period, as well as with an excellent hypoallergenic source of nutrition.

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Mucocutaneous Lymph Node Syndrome

MUCOCUTANEOUS LYMPH NODE SYNDROME (MLNS) is a recently recognized disease of unknown etiology which has a mortality of 1 to 2 percent. It has occurred with increasing frequency in Japan and is now being recognized in many countries throughout the world, including the United States. It is characterized by fever which persists for more than five days; an erythematous skin eruption; conjunctival congestion; dry fissured red lips; red tongue, palms, and soles; nonpurulent lymphadenopathy; diarrhea; arthralgia and aseptic meningitis. Additional striking features which occur less frequently include carditis; pericarditis; aneurysmal dilatation and thrombosis of coronary arteries; and sudden death. Fatal cases have features which are indistinguishable from infantile polyarteritis nodosa. In some surviving infants there is the transient appearance of coronary aneurysms which subsequently disappear. A few patients have recurrent disease but the vast majority appear to recover completely. Many cases mimic the Stevens-Johnson syndrome. MLNS may actually represent a subdivision of the Stevens-Johnson syndrome as well as one subdivision of infantile polyarteritis nodosa. It has many features of an infectious process and the possibility that the etiologic agent may be viral or rickettsial is currently under investigation in a number of laboratories.

During a collaborative investigation of Japanese children with the syndrome in association with Dr. Kusakawa it was found that early in the course of MLNS there is a 3- to 4-fold elevation of serum IgE level which peaks at 7 to 12 days after onset of the disease and declines over the ensuing 30 to 60 days. This suggests an element of hypersensitivity in the disease, and the vasculitis which commonly occurs suggests a similarity to serum sickness. There is also a significant elevation of total serum IgM but the peak is smaller and later than that of IgE. It is the first nonparasitic acute febrile disease which has been shown to be regularly accompanied by an elevation of serum IgE level and it is the first disease in which a marked IgE response has been shown to precede a significant IgM response. It appears that in patients who have recurrence of the disease the IgE level becomes elevated again, and in patients who have persistent active coronary arteritis IgE levels remain high. Serial IgE levels, therefore, may prove of diagnostic value and help in recognizing continued disease activity.

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The Hazard of IgE Mediated Allergic Reaction After Blood Transfusion

RECENT REPORTS have confirmed the potential risk to infants of passively transferred IgE following exchange transfusion. A single unit of blood or plasma containing a high titer of IgE or an exchange transfusion with blood containing normal amounts of IgE results in a 70 percent uptake by basophils and mast cells throughout the body within 24 hours. By 48 hours maximal fixation has occurred. The skin fixed IgE is shown to persist in significant quantities for at least 31 days. Consequently, the IgE infused in conjunction with a transfusion can place an infant at risk of an anaphylactic reaction for at least one month.

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